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SHORT REPORTS

An In Vivo Assessment of Physiological Arousal in Posttraumatic Stress Disorder

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The present study measured physiological function (heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP], sublingual temperature, and respiration rate) in a nonresearch setting—the medical triage area of a large Veterans Affairs Medical Center while patients were awaiting physical examination. Subjects were 32 Vietnam veterans with combat-related posttraumatic stress disorder (PTSD) and 26 Vietnam-era veterans with no combat experience. Results indicated that PTSD veterans had significantly higher HR, SBP, and DBP, but not sublingual temperature or respiration rate. These data support the position that individuals with PTSD do indeed demonstrate higher levels of cardiovascular arousal across settings.

Elevations in physiological arousal in individuals with posttraumatic stress disorder (PTSD) have received considerable attention both in the distant past (Kardiner, 1941) and more recently. From the studies conducted to date, several conclusions can be drawn. First, compared with nonveterans and veterans without PTSD, Vietnam veterans with PTSD are physiologically hyperreactive to combat-related stimuli (Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Gerardi, Blanchard, & Kolb, 1989; Malloy, Fairbank, & Keane, 1983; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Second, physiological reactivity is not present when PTSD patients are exposed to control (noncombat) stimuli. Third, PTSD patients can be physiologically discriminated from combat-exposed veterans with an anxiety disorder other than PTSD (Pitman et al., 1990) and this reactivity is related to scores on psychometrics that measure the severity of PTSD (Orr et al., 1990).

The primary limitation of the findings on psychophysiological arousal in PTSD is that all studies have been conducted within research laboratories, limiting the extent to which we can generalize from the findings. Recent conceptual models of PTSD have indeed incorporated the findings on physiological reactivity as fundamental features of the disorder (e.g., Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Foa, Steketee, & Rothbaum, 1989; Keane, Zimering, & Caddell, 1985). Thus, studies empirically documenting the existence of physiological hyperarousal in PTSD subjects in nonlaboratory settings would appreciably improve our understanding of the phenomenology of this disorder. It may also help us to understand the possible health consequences of PTSD that Blanchard (1990) and others have predicted might arise as a function of the aberrant psychophysiological arousal that accompanies PTSD.

Data for this study were obtained in a review of medical records from a sample of known cases of combat-related PTSD and from an era-, age-, race-, and sex-matched comparison group of noncombat veterans. Measures of physiological functioning were taken while patients were triaged in medical clinics. We hypothesized that PTSD subjects would manifest heightened psychophysiological arousal across response channels when compared with the non-PTSD veteran comparison group.

Method

Subjects

Subjects in this study were 32 Vietnam veterans with PTSD and 26 Vietnam-era veterans (medical) who did not serve in Vietnam. Forty PTSD subjects were randomly selected from records in the National Center for PTSD at the Boston Veterans Affairs (VA) Medical Center. Thirty-nine medical patients were randomly selected from the outpatient roster of the facility. A research technician examined each subject's medical record to extract the most recent Form 10-10m, a document completed by medical triage nurses when medical or psychiatric help is requested. Of the 32 PTSD subjects, 19 had requested care for physical problems and 13 for psychological problems. All PTSD subjects had been previously assessed by a clinical psychologist in the center. Assessments consisted of several hours of structured interviews, including the Boston PTSD Interview and Structured Clinical Interview

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Table 1
Reasons Subjects Were Excluded From Study

Reason	PTSD group	Medical group
Alcohol/drug use	6	3
Sympathomimetic medications	1	1
Pain	0	5
Injury	1	0
Illnesses	0	4

Note. PTSD = posttraumatic stress disorder.

for *DSM-III-R* (SCID), psychometrics (Mississippi Scale for Combat-Related PTSD [Keane, Caddell, & Taylor, 1988]; the Minnesota Multiphasic Personality Inventory (MMPI) [Hathaway & McKinley, 1951]; and the PTSD scale of the MMPI), and psychophysiological methods. Keane, Wolfe, and Taylor (1987) provide a thorough description of this multimethod diagnostic approach. Following the assessment, each subject was presented at a case conference, and final diagnoses were determined by consensus of the National Center staff (at least five doctoral-level psychologists and a psychiatrist).

The medical group consisted of male Vietnam-era veterans who had served in the military during the Vietnam war, but not in Vietnam. Because subjects in this group had never served in a combat zone, by definition they could not have combat-related PTSD. These veterans were coming to the Medical Center for medical (i.e., not psychiatric) services. Moreover, their medical records did not contain psychiatric diagnoses. The two groups did not differ in age (PTSD M = 39.3, SD = 3.8; medical M = 39.9, SD = 3.5), $F(1, 58) = .372$, $p = .54$, or racial composition, $\chi^2(2, N = 56) = 3.65$, *ns*.

Physiological Data

All physiological data were obtained from the last screening examination conducted by the triage nurse in the outpatient area of the hospital. These physiological data are part of the routine initial examination in triage. The triage nurses had access to subjects' VA medical records and were possibly aware of subjects' medical and psychiatric diagnoses. Yet, because this study was retrospective, nurses were unaware that these basic health data were to be examined for research purposes, thus minimizing the potential for systematic measurement bias. The dependent variables were heart rate (HR), systolic and diastolic blood pressure (SBP and DBP), sublingual temperature, and respiration rate. Heart rate and blood pressure were obtained with a Physio Control Lifestat 100 (Rowayton, CT), which is checked for accuracy at least once every 6 months by the Medical Center's Biomedical Engineering Service. Time intervals between the diagnostic procedure and the completion of the form ranged from 4 months to 2.5 years.

Exclusions

Subjects were excluded from the study if they presented with conditions that might affect baseline physiology (e.g., acute medical illness such as myocardial infarction or stroke, acute pain, use of beta-blocking agents, alcohol or drug intoxication). For these reasons, 8 subjects were excluded from the PTSD group and 13 from the medical group. See Table 1 for a complete listing of exclusions from the study.

Results

Analyses of the data indicated that the groups differed on several physiological measures. The PTSD group had significantly higher heart rate (PTSD M = 89 beats per minute [bpm], SD = 17; medical M = 78 bpm, SD = 9), $F(1, 56) = 8.19$, $p < .01$; systolic blood pressure (PTSD M = 133 mm, SD = 15; medical M = 124 mm, SD = 14), $F(1, 56) = 4.72$, $p < .05$; and diastolic blood pressure (PTSD M = 88 mm, SD = 12; medical M = 79 mm, SD = 11), $F(1, 56) = 7.95$, $p < .01$. The groups did not differ on sublingual temperature (PTSD M = 98.5, SD = .76; medical M = 98.4, SD = .84), $F(1, 56) = .39$, *ns*, or respiration rate per minute (rpm; PTSD M = 17.6 rpm, SD = 2.65; medical M = 18.7 rpm, SD = 2.48), $F(1, 56) = 2.36$, *ns*. In addition, there were no statistically significant differences on these physiological variables for subjects in the PTSD group who were seeking medical services (PTSDM) versus psychiatric (PTSDP) services: HR (PTSDM M = 90.2, SD = 18.7; PTSDP M = 88.8, SD = 13.7), $t(30) = 0.24$, $p = 0.81$; SBP (PTSDM M = 129.9, SD = 17.5; PTSDP M = 133.1, SD = 12.0), $t(30) = -.57$, $p = .43$; DBP (PTSDM M = 87.5, SD = 14.2; PTSDP M = 87.2, SD = 10.5), $t(30) = 0.06$, $p = .95$.

A separate analysis was conducted to more fully explore the possible effects of prescribed medication on cardiovascular functioning. Because this was a retrospective study, neither urine nor plasma level of medications was available. Consequently, the precise levels of prescribed medication are impossible to determine, but veteran psychiatric patients are generally known to have poor compliance. Nonetheless, one-half of the PTSD subjects (n = 16) were prescribed various psychoactive medications, and we examined, using a t test, differences between these subjects and the remainder (n = 16) on heart rate, systolic blood pressure, and diastolic blood pressure. The group with prescribed medications (i.e., tricyclic antidepressants, benzodiazepines, etc.) did not differ from those for whom medications were not prescribed, $ts(30)$ = .89, .16, and .07, respectively.

Discussion

Previous research suggests that Vietnam veterans with PTSD may have persistent arousal across psychophysiological systems. These findings have significant implications for conceptual models of PTSD, the psychopathology associated with PTSD, and cardiovascular morbidity if found to be generalizable beyond the laboratory. The present study is the first to demonstrate this arousal in a nonlaboratory setting. In particular, we found that measures of cardiovascular arousal (HR, SBP, and DBP) were elevated for the PTSD group, whereas measures of sublingual temperature and respiration were equivalent for the two study groups. All subjects were exposed to ostensibly the same situational stressor.

To understand the PTSD subjects' differential response to a stressor, we posit that seeking medical care of any type stimulates a trauma network that is fundamentally related to PTSD (cf. Litz & Keane, 1989). Fears of bodily injury and illness and the lack of personal control in stressful situations are central tenets of our previous conceptualization of the disorder (Keane et al., 1985). Visiting a medical facility for medical or psychiatric reasons can instigate these cognitive processes and lead to the heightened physiological arousal observed in this study.

These findings must be delimited by the recognition that measurement occurred in the context of help-seeking, a complex experience certainly characterized by elevations in stress

for all. The observation of increased cardiovascular reactivity among PTSD patients in this study supports the proposition that they have relatively greater reactivity across different stressors and that this may place them at an enhanced risk for developing cardiovascular disease (Blanchard, 1990). One further limitation to the findings of this study is the observation of increased variance in the heart rate of the PTSD group. With nearly twice the standard deviation of the medical group, this elevated variance appears to reflect true variability about the mean for the PTSD group, and it may be a function of the frequently observed psychological and physiological comorbidity of help-seeking PTSD patients (Keane & Wolfe, 1990).

McFall, Veith, and Murburg (1992), in their laboratory study of basal adrenergic functioning, found that tonic levels of HR, SBP, and DBP were not elevated when compared with a non-PTSD group. Subjects in their study did not anticipate exposure to trauma-related stimuli, which removes this confound from previous laboratory research. It also highlights the importance of categorizing the present study as one of reactivity to a naturally occurring, but distinctly not trauma-related, stressor.

Future research utilizing continuous monitoring of psychophysiological arousal and reactivity (e.g., using ambulatory monitors) would be a welcome addition to research in this area and would provide evidence to further bear on the nature and extent of the hyperarousal and hyperreactivity observed to date in combat-related PTSD. Studies on psychophysiological arousal in PTSD that is not combat-related would also increase available knowledge on the disorder more generally.

Although the present findings are clearly limited to combat-related PTSD, these results do demonstrate the powerful component that psychophysiological arousal plays in PTSD as initially explicated by Kardiner (1941). At that time he referred to the disorder as a physioneurosis on the basis of his personal observations of psychophysiological abnormalities. The identification of structural and physiological substrates in both the central and peripheral systems responsible for these arousal patterns is a potentially fruitful direction for future research. The delineation of such parameters would provide targets for clinical interventions. For now, interventions emphasizing the direct modification of these patterns of arousal and reactivity are recommended (e.g., Keane, Fairbank, Caddell, & Zimering, 1989).

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